of protein, 146 gm. of fat, 250 gm. of carbohydrate, 2800 calories. The subject had been in coma at least once.

Case 36. Secretin test: 3-3-41. Urine: 3-2, sugar ++; 3-3, sugar ++; 3-4, sugar +. Fasting blood sugar: 2-14, 150 mg. per cent; 3-9, 272 mg. per cent. Insulin: 3-2, 55 units of regular. Diet: 100 gm. of protein, 201 gm. of fat, 200 gm. of carbohydrate, 3000 calories.

Case 13. Secretin test: 2-12-40. Urine: 2-11, sugar +; 2-12, sugar trace; 2-13, sugar ++. Fasting blood sugar: 2-9, 270 mg. per cent; 2-12, 232 mg. per cent. Insulin: 2-11, 30 units of regular and 40 units of protamine-zinc insulin. Diet: 60 gm. of protein, 150 gm. of carbohydrate, 1400 calories.

DISCUSSION

DR. LEMUEL C. McGEE (Wilmington, Del.): I think any approach to the secretion of the small bowel which should give us information regarding the electrolyte or enzyme content, is quite important.

I was impressed by the change which was found in those older diabetics of the two groups, the bicarbonate content.

Several years ago Carr and Abbott, using the Miller-Abbot tube in intubation, demonstrated a certain reciprocal relationship between bicarbonate and the chloride content. They represent the two chief negative ions.

Subsequently, in some work which was published by Dr. Emery and myself, and by Dr. Hastings and myself, it was demonstrated that these two negative ions not only were reciprocal, as previously indicated by Carr and Abbott, but maintained a fairly constant pH in the upper small bowel.

I would question the advisability of attaching too much significance to a low bicarbonate content unless the chloride was determined at the same time. If he has the chloride observations, I should be interested to hear his comment upon this reciprocal relationship of which I am sure he is aware.

DR. H. MARVIN POLLARD (Ann Arbor) (closing the discussion): In answer to Dr. McGee's question, we did not determine the chloride content. We did determine the free acid in the gastric contents, but there was no determination made of the chloride content in the duodenum.

The Gastric Mucosa, "Gastritis" and Ulcer

By

STEWART WOLF*

and

HAROLD G. WOLFF

NEW YORK, NEW YORK

STUDIES have been pursued on a 57 year old subject with a large permanent gastric fistula 3.5 cm. in diameter surgically produced in 1895 because of benign stricture of the oesophagus. The mucosa lining the stomach has been examined by direct inspection and gastroscopically, and has been found to be of normal appearance. Likewise secretory and motor functions have been studied and found to be undisturbed (1). The present communication deals with an investigation of changes in appearance of the gastric mucosa of this individual after various stimuli.

Since the advent of gastroscopy there has been much interest in certain deviations from the usual appearance of the stomach mucosa called "gastritis." The changes have been classified by various observers and morbid symptoms have been attributed to some of them (2, 3, 4).

Beaumont (5) was among the first to describe the clinical appearance of "gastritis." He called attention to unusual redness or pallor of the mucous membrane of St. Martin's stomach accompanied by small lesions which appeared pustular or more commonly greyish crusts which he called "aphthous spots." His descriptions are not detailed enough to enable the present day reader to visualize exactly what he saw. In fact, he usually merely noted that the stomach was of a "morbid appearance," and that most often after what he considered excessive eating or drinking of spiritous liquors on the part of his subject.

The "aphthous spots" which Beaumont noted have already been discussed elsewhere (1), and it appears possible that he referred to flecks of rolled up precipitated mucus which adhered in places to the lining of the stomach.

The occurrence of pustular lesions in the mucous membrane of the stomach has never been confirmed by subsequent observers and it is likely that in the face of the unsatisfactory lighting conditions and other difficulties of seeing clearly into his subject's stomach, with which Beaumont had to contend, he misinterpreted the appearance of some of these mucus flecks as pustular. It is noteworthy that nowhere does he mention actually having recovered pus from one of them.

As for the "abnormal" redness or pallor of the mucosa which Beaumont observed, we have shown elsewhere (1), that profound color changes occur in the gastric mucosa in the absence of disease and in association with varying day to day conditions which must be regarded as normal.

It appears, then, that there is some doubt as to whether color changes and the appearance of localized spots on the stomach lining represent disease or "gastritis" or whether they should be looked upon as incidental variations within the range of "normal."

This same question arises among gastroscopists today (6, 7, 8). There is much evidence to support the notion that small hemorrhagic lesions and so-called "pigment spots," regarded by many as signs of disease (2, 3, 4), are in reality artefacts due to the trauma of the gastroscope itself or to the suction applied to the wall by the stomach tube in making the preliminary gastric analyses. Ruffin (12), for example, has shown that the application of suction through a

*National Research Council Fellow in the Medical Sciences.
From the New York Hospital and Departments of Medicine and Psychiatry of the Cornell University Medical College, New York, N. Y.
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Rehfuss tube in the process of obtaining gastric juice from the stomach sucked the mucosa against the holes in the tube and caused small submucosal and intramucosal hemorrhages. After several minutes these spots became black and appeared identical to the lesions described by many as "pigment spots."

Another change which has been considered of special significance in the diagnosis of "gastritis" is the size of the rugae in the stomach as they appear through a gastroscope. Ruffin (6) has shown that their

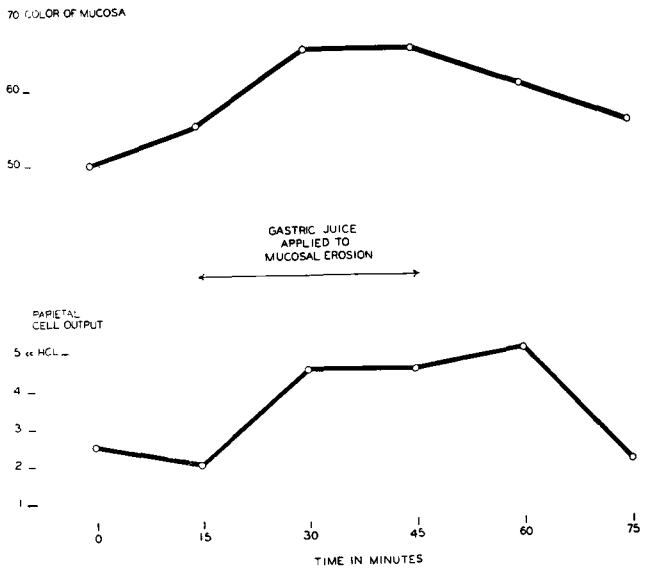

Fig. 1. The acceleration of acid production following contact of gastric juice with an eroded mucosa.

size may be a function of the quantity of air used in inflating the organ to provide an adequate view. With relatively large amounts of air, but not more than may be used during the course of ordinary gastroscopy, he found that the stomach lining could be flattened out entirely, so that it simulated the appearance of so-called atrophic gastritis. These observations we have confirmed in our subject as mentioned elsewhere (1).

"The Hyperfunctioning Stomach" and "Hypertrophic Gastritis." It has been pointed out in another publication that a hypersecreting stomach was always hyperaemic, turgid, engorged. This was true regardless of whether the accelerated secretion occurred in response to the stimulus of food, alcohol, histamine or to certain emotionally charged situations (10). When such a state of engorgement and hyperfunction was intense and prolonged, the gastric mucosa assumed the characteristics of what is known to gastroscopists as "hypertrophic gastritis." The folds became thick, red and succulent. The slightest blow with the side of a glass rod, or stroking with a dry gauze, resulted in the appearance of small hemorrhagic spots and erosions. Frequently these minute hemorrhages occurred spontaneously, following vigorous contractions of the stomach wall. The contractions themselves caused pain when they were of a sufficient magnitude, but not when the mucosa was pale and non-oedematous (11). When this hyperfunctioning, engorged condition of the stomach was prolonged in our subject, he frequently complained of abdominal discomfort and pain.

Comment. It is clear from above that many of the signs of "gastritis" may be encountered in the normally functioning stomach or may be reproduced therein by minor traumata associated with instrumentation. To none of these changes discussed is the term "gastritis" altogether properly applied, since there was no indication that actual infiltration of inflammatory cells occurred. The picture of "hypertrophic gastritis," for example, was found to be induced by vasomotor changes resulting in hyperaemia and congestion of the mucosa. These changes were often transitory, disappearing within an hour or two, a time too short for the subsidence of a true inflammatory process.

The fact that the congested mucosa was especially susceptible to injury, however, suggested that vascular engorgement might predispose to the development and persistence of erosions and changes secondary to inflammation. Furthermore, sustained hyperaemia of the gastric mucosa assumes added significance, since it was found to be accompanied by symptoms of abdominal discomfort and pain.

The Resting Gastric Mucosa. In view of the special characteristics attributable to the mucosa during gastric hyperfunctioning, it seemed desirable to examine the behavior of the pallid resting gastric mucosa.

Method. The mucosa was examined after a fast of 12 or more hours. In each case the stimuli were applied to folds of mucous membrane which ordinarily lay within the cavity of the stomach, but which were forced out through the stoma for the occasion by having the patient increase his intra-abdominal pressure. As noted elsewhere, this region of the stomach lining was of altogether healthy appearance. Many of the tests were also carried out on the collar of mucosa which always protruded through the stoma, and a few were made on remote parts of the stomach lining. In the latter group, observations of the membrane were made through a gastroscope.

After observing and, in many instances, photographing the normal appearance of the mucous membrane, the stimulus was applied. The area was then observed continuously for one hour. Effects which persisted for a longer period were checked at intervals until they had disappeared.

The stomach was allowed to rest for from 2 days to

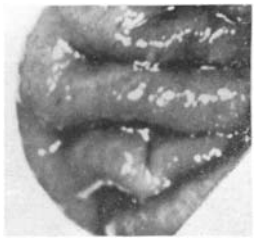

Fig. 2. Normal mucosal folds (actual size).

a week between these particular experiments, and they were undertaken only when the gastric mucosa appeared relatively pale and inactive.

OBSERVATIONS

Sudden Mechanical Trauma. When the mucosa was struck a sharp blow with the side of a glass rod, the area struck became blanched and depressed within one second. It remained so from 1-5 seconds, depending on the force of the blow. Following this there occurred

a slight transitory hyperaemia in the same region, which lasted for 3-10 seconds.

Comment. This effect has been observed before in dogs by others (13), who concluded that the trauma brought about a reflex contraction of the muscularis mucosae, which squeezed the blood from the mucosa for a few seconds.

Continuous Mechanical Irritation. For 15 minutes an area of 2 sq. cm. was rubbed gently with the blunt end of a glass rod. Within 5 minutes the area had become slightly red and an obvious acceleration of mucus secretion had occurred. The mucus was thick and transparent and clung tenaciously to the wall of the stomach. A drop of Toepfer's solution allowed to fall on the region turned distinctly yellow, indicating that its pH was higher than 4.3.

Negative Pressure. The stomach mucosa was sucked up against the hole in the side of a soft rubber catheter by the moderate negative pressure exerted by a 4 cm. column of mercury for 1 minute. At that point a small, slightly-elevated purpuric spot, the size of the hole in the tube, appeared. After contact with the acid contents of the stomach for 15 minutes, the spot turned brown and then black. It thus had precisely the appearance of "pigment spots" in the stomach described by gastroscopists. The force of the negative pressure which caused this lesion was far less than that usually applied with a syringe in the course of a routine gastric aspiration which regularly precedes gastroscopy.

In order to measure the force ordinarily applied during gastric analysis the syringe was connected directly to a mercury u-tube manometer while a technician exerted the usual moderate tension on the plunger. The average negative pressure during 10 trials was 10 cm. of mercury.

Abrasion of the Gastric Mucosa. The Protective Power of Mucus. Crystals of sodium chloride were sprinkled on the gastric mucosa and were then gently rubbed across its surface with the finger. Small linear hemorrhagic lines were produced where the sharp crystals had scratched the surface. A prompt accumulation of mucus was observed in the injured area. The appearance of the stomach lining elsewhere was not changed and no extra mucus secretion was observed except in the injured area. Here the acceleration of mucus secretion was estimated at 3 to 6 fold. No local oedema or other evidences of inflammation were observed.

The following morning, approximately 24 hours later, all but a few of the larger lesions had disappeared. Here one saw a scale of opaque precipitated **mucus adhering firmly to the injured area. Its surface was spotted with blackened blood pigment.**

This lesion had precisely the appearance of what is spoken of by some gastroscopists as superficial gastritis (2, 4).

48 hours after the lesion was inflicted, all evidence of abnormality had disappeared. This minor injury to the gastric mucosa was not accompanied by digestive complaints of any sort. The patient ate 2 hours after it was inflicted, and subsequently at his usual intervals. The stomach digested the food and emptied in the usual time.

Effects of Chemical Trauma. Acids and alkalis, drugs commonly taken by mouth, and condiments were applied directly to the stomach lining. Each substance was rubbed very lightly on an area one centimeter square. Another area of similar size removed from the first site was selected as a control. This region was also rubbed lightly. Both were observed continuously for an hour. The reactions produced by irritating substances were classified in the following manner: A slight erythema within the limits of the test area was called a 1+ reaction. Moderate erythema within these limits was termed 2+. A 3+ was applied to erythema which extended beyond the one centimeter square, and 4+ was used to indicate an inflammatory reaction extensive enough to cause evidence of oedema of the mucous membrane. Below is a tabulation of the results obtained with the substances tested:

Drug	15 Min.	½ Hr.	1 Hr.
Alcohol 20%	0	0	0
Alcohol 50%	0	0	0
Alcohol 100%	0	0	0
Histamine 1%	0	0	0
Acetyl salicylic acid (Powd. pill)	0	0	0
Sulfanilimide	0	0	0
Sulfapyridine	+	++	++
Sulfathiazole	0	0	0
Sulfaguanidine	0	0	0
Sulfadiazine	0	0	0
Ammonium chloride	0	0	0
Digitalis	0	0	0
Quinidine	0	0	0
Glucose 50%	0	0	0
Mustard (1-30 susp. in water)	+	++	++
Hydrochloric acid 1 N	+	++	++
Sodium hydroxide 0.1 N	+	++	++

These same agents were similarly applied to an area on the volar surface of the forearm. The 50% and 100% alcohol produced moderate local erythema. The mustard, hydrochloric acid, and sodium hydroxide produced swelling, tenderness and vesiculation in addition to erythema. These effects were far more intense than the designated 4+ reaction.

Comment. The failure of strong irritants and corrosive agents to cause more than a slight-to-moderate erythema in the gastric mucosa, while they caused **marked reaction with destruction of tissue when applied in similar concentration to the skin, is striking. It indicates that the cells lining the stomach are endowed with some special protection against chemical** injury. The neighboring esophagus is not so well protected. Irritants brought into contact with the latter are well known to cause inflammation with pain and occasionally stricture.

Presumably the special protection is afforded by the thick layer of tenacious mucus which is adherent everywhere to the stomach lining and which is elaborated in increasing amounts in response to physical and chemical stimuli, thus protecting the membrane from significant injury.

Properties of Mucus. When a drop of Toepfer's solution was allowed to fall on the exposed collar of the mucous membrane or even on the stomach wall within the stoma, it failed to turn red until it came in contact with an accumulated pool of gastric juice. When this test was made, a specimen had just been poured out containing 65 units of free acid, and all parts of the stomach wall including the exposed collar were moist with that secretion. A drop of phenolphthalein was applied in like manner and this, too, failed to become red. Thus it was concluded that the pH of the surface of the stomach wall lay between 4 and 7, although it enclosed a juice of less than pH 2.

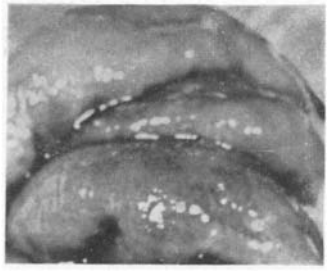

Fig. 3. Engorgement of mucosa accompanying emotional conflict (same scale).

Thus the layer of mucus which clings everywhere to the stomach lining is sufficiently alkaline to maintain the surface of the gastric mucosa in a relatively neutral environment, despite the high concentration of acid in the gastric juice which it encloses.

In addition to its capacity to neutralize, mucus displayed another characteristic which makes it an effective insulator for the cells lining the stomach. This was demonstrated in the following way.

1 N hydrochloric acid was allowed to fall upon the gastric mucosa at the rate of 12 drops a minute. At once the acceleration of the output of mucus was apparent, and the mucus which the drops of acid touched became grey and opaque. This layer was pulled away with considerable difficulty from a small area. The grey, membranous flakes thus obtained were found to be relatively insoluble in gastric juice, in 0.1 N hydrochloric acid and even in 1.0 N hydrochloric acid.

To test this protective power, an attempt was made to deprive a part of the gastric mucosa of its mucus covering and then to subject it to irritating stimuli.

Production of Gastritis After Attempts to Remove Covering of Mucus. 1.0 N hydrochloric acid was allowed to fall upon the exposed gastric mucosa drop by drop as outlined above. Within 2 minutes a thick layer of greyish opaque mucus had appeared over the area exposed to the acid. The rate of application of the drops was increased from 12 to 20 a minute, while the accumulated mucus was sucked away through a pipette. Within 5 minutes the mucosa beneath became moderately reddened and oedematous. The 1-30 mustard suspension was then applied and its effect observed. Within 5 minutes the redness and oedema was further accentuated and very minute bleeding points became evident as pin-point black specks on the mucosa where drops of hemoglobin had been altered by the hydrochloric acid present.

Mechanical trauma were also applied to the gastric mucosa in its state of induced inflammation. A sharp blow with a glass rod which formerly resulted in a transitory blanching and subsequent reactive hyperaemia now induced minute bleeding points. Rubbing with the blunt end of a glass rod also caused hemorrhage, and as pointed out in another publication (11), these mechanical stimuli applied to such an area caused pain.

Acceleration of Acid Output by Contact of Gastric Juice with Minute Erosions. Two of the small hemorrhagic lesions described in the preceding paragraph were kept in contact with gastric juice with a titratable total acid of 90 for ½ hour. Mucus accumulated rapidly in the region, but it was removed at frequent intervals by suction through a small glass tube, and the acid gastric juice was then reapplied to the bare mucosa. A sharp acceleration of acid secretion and concomitant hyperaemia of the whole stomach mucosa occurred and persisted for ½ an hour after the submersion of the hemorrhagic lesions was discontinued (Fig. 1).

Comment. In this phenomenon may lie an explanation of the persistent hyperacidity regularly encountered in persons suffering from "gastritis" and peptic ulcer. The fact that the base of the ulcerated lesions which is constantly bathed in acid gastric juice effects a stimulation of acid secretion indicates that afferent impulses subserve this reflex without sensation resulting. It is likely, however, that pain would follow an adequate chemical stimulus (14).

The Effect of Gastric Juice on an Area of Mucosa Lacking in Mucus. The most peripheral edge of the collar of mucosa which lay exposed on the abdominal wall lacked adequate protection owing to defective formation of mucus in this region. A small erosion which occurred on this peripheral edge was exposed continuously to the digestive action of gastric juice for 4 days. During the first 24 hours the denuded surface increased in size. It bled intermittently. At the end of 4 days it exhibited the typical punch-out appearance of a chronic peptic ulcer with well-defined edges and a granulating base. It measured approximately 4 mm. in diameter, 1 mm. in depth and was growing rapidly (Fig. 2). Traction or pressure on this lesion resulted in pain of a dull, gnawing character, which was localized in the region of the lesion itself. Throughout the 4 day period the whole mucosa was relatively engorged, and the rate of acid secretion was significantly elevated.

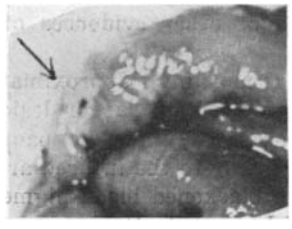

Fig. 4. Mucosal erosion (same scale).

At the end of 4 days because of the hazard to the subject it was felt that the experiment could not be allowed to continue.

The ulcer and surrounding area were covered with a protective petrolatum dressing. Within 3 days com-

plete healing had taken place, leaving no trace of the lesion behind.

DISCUSSION

It is clear that gastric juice is quite capable of attacking and digesting the mucous membrane of the stomach. It does not do so under normal circumstances because it does not gain access to it. Neither do other irritating and corrosive substances taken as articles of diet, because the lining of the stomach is normally covered by an efficient insulation in the form

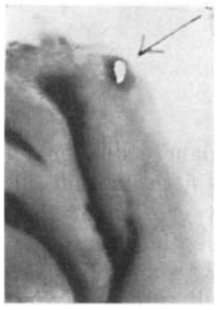

Fig. 5. Punched-out ulcer resulting from prolonged contact of gastric juice with an area of mucosa inadequately protected by mucus (same scale).

of a protective covering of tenacious, viscous, alkaline mucus. The latter combines with the acid in its immediate vicinity and thus maintains the cells of the gastric mucosa in a relatively neutral chemical environment. It effectively diverts the force of any mechanical trauma or abrasive which may brush by, by presenting a slimy, mucinous surface. Finally, in the presence of strong acid it precipitates and forms a relatively insoluble membranous shell over the delicate mucosal cells. Once the vast protective powers of mucus are overcome, however, and the digestive juices have attacked and eroded the surface of the mucous membrane, a vicious cycle is set up since the acid gastric juice in contact with a denuded region induces further acid secretion.

Chronic ulceration then is the resultant of the interplay of forces protective and destructive. If a minor erosion can be effectively covered with mucus and the hyperfunction of the stomach subsides spontaneously or can be made to subside by the ingestion of fat or the administration of drugs, healing takes place quickly and uneventfully. If, on the other hand, hyperaemia and hypersecretion are sustained by a stimulus which overwhelms the inhibitory influence of fat or drugs, the susceptibility of the mucosa to injury is enhanced and tissue damage proceeds unchecked, resulting in ulceration.

SUMMARY

The healthy gastric mucosa varied in appearance within a wide range. When the rate of acid production by the parietal cells was relatively slow the mucosa was always comparatively pale and in this state relatively resistant to injury unless the continuity of its protective covering mucus was interrupted.

Accelerated acid production and motor activity were always accompanied by hyperaemia and engorgement of the mucosa. When vascular engorgement was prolonged the rugae became intensely red, thick and turgid presenting the picture of what has been called "hypertrophic gastritis." In this state the mucosa was unusually fragile, haemorrhages and small erosions resulting from even the most minor traumata. Lowering of the pain threshold occurred and symptoms were often associated with this condition. Thus the difference between hyperfunction in the stomach and "hypertrophic gastritis" was seen to be mainly one of degree. Continued exposure of a small erosion to the digestive action of gastric juice for 4 days resulted in a peptic ulcer.

CONCLUSIONS

1. Undue and prolonged acceleration of acid secretion in the stomach, however provoked, resulted in hyperaemia and engorgement of the mucous membrane resembling hypertrophic gastritis.
2. The mucosa in this state was unusually susceptible to injury, and even the most trifling traumata resulted in hemorrhages and small erosions.
3. Ordinarily the mucosa was protected from injury by an effective coating of mucus. Loss of this protection in the face of minor traumata led to oedema, inflammatory changes, erosions and hemorrhages.
4. Contact of acid gastric juice with a denuded surface induced further hyperaemia and acceleration of acid secretion.
5. Prolonged contact of acid gastric juice with a minor erosion resulted in the formation of a peptic ulcer.

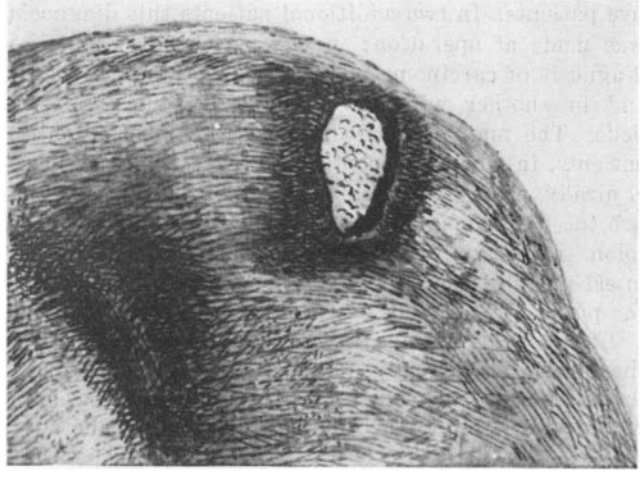

Fig. 6. Drawing of ulcer (enlarged five times).

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Peptic Ulcer in the Aged; a Clinical and Post-Mortem Study

By

JACOB MEYER, M.D.

and

OTTO SAPHIR, M.D.

CHICAGO, ILLINOIS

IT is generally believed that peptic ulcer in the aged is uncommon. Recently, however, Mulsow (1) reviewed the literature on peptic ulcer in the aged, and stated that 10.5% of 4,079 patients were above the age of 60. The concept generally held is that when peptic ulcer occurs in the aged, it is chronic, of many years duration, and that the hemorrhage and perforation which occur are complications of an old peptic ulcer.

This study is a report not only of the occurrence of chronic peptic ulcer in the aged, but also of the occurrence of recent acute peptic ulcers in the sixth and later decades of life. Such acute ulcers constitute an unexpected and often unrecognized complication of many diseases and are often the cause of death because of hemorrhage or perforation. Occasionally these acute ulcers became chronic.

This communication comprises the study of 16 patients. The youngest was 60 years old, the oldest, 83. Six were between 60 and 69 years old; seven between 70 and 79, and three over 80 years. There were 12 males and four females. Two patients were negroes.

The clinical diagnosis of peptic ulcer was made in five patients. In two additional patients this diagnosis was made at operation: in one patient in whom the diagnosis of carcinoma of the stomach had been made, and in another with the pre-operative diagnosis of ileus. The main clinical diagnosis of the other nine patients, in whom the peptic ulcer was not recognized clinically, were: carcinoma—of the bronchus; of the prostate; of the sigmoid colon; and of the stomach, or colon, respectively; cholecystitis with cholelithiasis; intestinal obstruction in two; perforated viscus; and cor pulmonale.

The clinical findings in the five patients in whom the ulcer was correctly diagnosed, were quite characteristic and corresponded with the findings in patients with peptic ulcer in the younger age group. The ages of these patients were 60, 62, 74, 79 and 81 respectively. Post-mortem examination disclosed that one patient had a penetrating gastric ulcer; another a recent gastric and duodenal ulcer; the third had two duodenal and two gastric ulcers, one of which had perforated and produced a diffuse peritonitis; the fourth had a penetrating ulcer in the duodenum with erosion of the pancreoduodenal artery and fatal hemorrhage. The fifth patient also died of hemorrhage produced by erosion of the left gastric artery by a penetrating peptic ulcer.

The following two short clinical abstracts may indicate that it is possible to recognize the onset of acute peptic ulcer in patients who are 60 years old or older, and who never have had a previous story of epigastric distress.

A 61 year-old white female developed epigastric and precordial distress. There was no complaint of discomfort in her earlier life. X-ray examination revealed a gastric ulcer on the lesser curvature. One year later she had a severe hematemesis, from which she recovered on simple medical management. Five years later she again experienced a severe hematemesis, the red blood corpuscle count being 1,300,000. She improved on medical treatment. An X-ray examination at this time showed a defect on the lesser curvature, and she was referred to the out-patient department.

Another patient, a male, at the age of 75 developed epigastric distress typical of ulcer. An X-ray examination revealed a duodenal defect. He was treated in the out-patient department for a duodenal ulcer and for diabetes. Eventually he developed a carbuncle of the neck, was admitted to the hospital, and died two days later. The autopsy disclosed a duodenal ulcer with erosion of the pancreoduodenal artery.

These two patients illustrate the fact that an acute ulcer may occur in people past sixty years of age, and that the ulcer may be recognized and treated as such. Mulsow has emphasized that gastric syndromes in the aged generally raise suspicions of carcinoma, and that the possibility of peptic ulcer is not even entertained. His patients had chronic ulcer of many years' duration.

Among clinicians the concept prevails that the occurrence of an ulcer syndrome in the aged is due to recurrence of symptoms of an old ulcer. The point is generally made that aged patients generally forget the history of previous gastric symptoms. Such statements are undoubtedly correct in some instances, but it has been our experience to find evidence of acute gastric and duodenal ulcers together with evidence of healed or chronic gastric and duodenal ulcers. Two cases may serve to illustrate such an occurrence.

*From the Departments of Internal Medicine and Pathology of Michael Reese Hospital, Chicago, Illinois.
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